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EVOLUTION BY MUTATION

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STUDENTS of Mendelian heredity have shown during the last seventeen years great reluctance in bringing their results to the bar of the evolutionary theory. They have hesitated, I think, because they felt that they had in hand a body of well-established information in regard to heredity which they did not want to compromise by applying to it the kind of procedure that in other fields of evolution is, even to-day, taken seriously.

The pressure on the Mendelians has come, then, from without rather than from within. But the Mutationists and the Mendelians are twin brothers because they appeal to the same scientific procedure, viz., analytical experimentation, and because both study the process of discontinuous variation. It has been more than insinuated, more than once and from more than one quarter, that the kind of unit characters that the Mendelians are studying has nothing to do with the kind of characters that evolution has to deal with. On what kind of evidence is this assertion based? Evidently it has its roots in the notorious fact that the Mutationist pays very little attention to the kind of a character that he uses in his work; the more extreme, the better, because he can the more easily identify it. But *he* knows (although the systematist tries to make it appear that he does not know) that between such extreme and even bizarre characters, and those that systematists themselves use for diagnostic purposes there is every gradation conceivable.

SPECIES AS GROUPS OF GENES

One of the most interesting ideas that De Vries brought forward in his mutation theory was that groups of "small species" or of varieties are made up of many common genes and differ in a relatively small number of genes. The genetic analysis of a group of smaller species would consist in finding out how the different genes were distributed amongst the members of this group. Phylogenetic relationship comes to have a different significance from the traditional relationship expressed in the descent theory; but this point of view is so novel that it has not yet received the recognition which we may expect that it will obtain in the future when relationship by common descent will be recognized as of minor importance as compared with relationship due to a community of genes while differences are due to different combinations of genes.

If related species have many genes in common they may be expected to produce at times the same mutants. In fact, it is not at all uncommon to find even in Mendelian literature such forms as albinos spoken of as though they represent the same mutation wherever it arises. Attractive as such a view appears, experience has shown that it is very unsafe to judge as to the nature of the mutation from the appearance of the character alone. Two different white-flowered races of sweet peas are known which give the wild purple-flowering pea when crossed, showing that they represent different mutations. Similarly, at least two recessive white races of fowls are known, as well as a third dominant white race. Three independent mutations have produced white birds. Whether albino mice, rats, rabbits, squirrels and guinea pigs have arisen through a mutation in the same common gene can not be determined because they can not be crossed to each other. When we consider that many factors may combine to produce a given pigmented animal and that a change in any one of them may affect the end result, it will be evident that the expectation would be against rather than for the conclusion that the same gene had changed in all cases. Only when it could be shown that a particular gene of the complex is more likely to change in a given direction than other genes of the complex would this interpretation become plausible.

There is some evidence in *Drosophila melanogaster* showing that the same mutation to white eyes has occurred several times, and the additional and all-important proof obtained that it is the same locus that has produced the white-eyed mutant. This may appear to give some slight support to the view that albino mutants appearing in other related species may be due to the same mutative changes but without additional evidence this conclusion is problematical.

In the mammals melanic individuals have been frequently described, but there is no direct evidence to show that they are due to the same change. In the roof rat there is a black type that is dominant to the gray of this race, while the black type of the Norway rat is recessive to the gray of that race. It seems probable that they are different mutations but not necessarily so.

Yellow in the mouse is dominant and lethal; two races of yellow rats are known, both recessive forms. The relation of yellow to black in mice is different from the relation of either of the yellows to black in the Norway rat. If the blacks are the same mutant the yellows are different, if either yellow of the rat is the same as the yellow of the mouse the blacks must be different, etc.

The uncertainty of reaching any conclusion in regard to the nature of the mutation from the appearance of the character of the mutant is excellently illustrated in such a group of mutants as that of the fruit fly, where a considerable number of cases are known in which mutants that are almost indistinguishable externally have been shown to be due to mutations in different parts of the germ plasm. There are five kinds of black mutants, three or more yellows and several eye colors that are practically indistinguishable. The evidence showing their difference is based on the results of crossing, where, as a rule (except, for example, for complete or incomplete dominants), reversion to the wild type occurs. In addition, the localization of the gene causing the modification shows them to be different.

The method of localizing genes opens up an opportunity for obtaining evidence in regard to like-mutants in related species that can not be crossed and a step forward in this direction has been taken by C. W. Metz for other species of the genus *Drosophila*. In a species, *D. virilis*, he has had 12 mutants appear and these fall into three groups of linked genes. Three of them, yellow, forked and confluent, resemble externally characters of *D. melanogaster*. Yellow and forked are sex-linked and look like the same characters in *melanogaster*. Confluent is like a second chromosome character of the same name in *melanogaster* in three respects: first, the structural similarity, second, that the character is dominant in both forms, and third, that it is, Metz thinks, lethal in homozygous state. The terminal position of yellow and the large amount of crossing over with forked are roughly speaking the same in both.

Even in this case further work is needed, first, because within the same species the occurrence of similar-looking characters due to different factors is known, *i. e.*, there are two genes for yellow color (yellow and lemon) in the first chromosome and in the same part of that chromosome, and second, because it is not to be expected that the number of crossovers would be identically the same between the same loci in different species, since marked variations are known within a single species.

Unless such species can be crossed the only convincing evidence that we can hope to get will be to establish the same *linear order* in the chromosome for several genes whose characters appear to be the same or similar.

IS THE DIRECTION OF MUTATION GIVEN IN THE CONSTITUTION OF THE GENE?

Whether we think of the gene as a complex molecule or as a quantity of material holding together through cell generations, it seems plausible that its mutations will be conditioned by its nature. If it is a molecule these changes may be strictly limited, numerous though the possible changes may be; but if it is only a definite "quantity" of something, the amount of its increase (plus) or decrease (minus) might *seem* less restricted, since any amount of increase or decrease might appear possible. We are left to pure speculation. Do the observed facts of mutation furnish any data on which to base even a guess? As has been said, we are scarcely warranted in drawing any conclusions from the nature of the character as to the nature of the gene, because, *a priori* at least, any change in the gene might cause an increased or decreased effect on the characters chiefly influenced by that gene. If, however, we boldly disregard this danger it may seem plausible that the building up of a complex molecule would be more likely to have a more limited range of possibilities than the rearrangement or degradation of that molecule. If this could be made probable, progress, in the sense of greater genic complications, would seem less likely to occur than degeneration. As hinted before, the recessive nature of the great majority of observed mutations might seem to harmonize better with this view. But we are too ignorant concerning these questions to make a discussion of them profitable.

Is the outlook any brighter if we take only quantitative changes in the gene as a basis for speculation? If the gene varies about a modal amount that accounts in part (for the environment also comes in here to complicate the effects on the character) for the degree of development of the character, it would seem to follow that the more the amount of the gene, the greater its effect on the character, and the less its amount, the smaller its effect. If we go further and suppose that nearly the maximum amount of a gene possible under the physiological conditions of the cell is more likely to be that maintained by selection in the wild species, it would follow that decreases in this amount are more likely to happen than increases, hence the more frequent occurrence of mutation by loss or recessive mutations. The facts concerning allelomorphic series indicate that the amounts "lost" (if the change is of this sort) are not progressive, but, so to speak, haphazard, and this, too, is consistent with the idea that such recessive changes may involve any amount of loss. Contrariwise, if the upper range in amount is near the maximum possible for the cell physiology, positive advance might take place more gradually and progressively, giving changes like those of orthogenesis claimed by paleontologists and some systematists to be the way in which species advance. This hypothetical situation is, however, uncertain in a high degree the moment one realizes that changes up and down of a character appear more often brought about now by one, now by another gene, rather than of the principal one, *i. e.*, the one on which, owing to a mutation, especial attention is focused. In fact, at present it is not even probable that progressive character changes are generally due to progress in the principal gene (except in multiple allelomorphs) and there is verifiable evidence in the only cases where really crucial evidence has been obtained that the changes are due to other genes.

It is true that when writers have brought forward evidence of continuous and progressive change in a character they have not concerned themselves with the analysis of the change in the germ plasm that has brought it about—in fact, in most of these cases the possibility of advance in a principal gene or of advance through modifying genes had not been appreciated or even understood. Paleontologists who have in the main been the strong advocates of orthogenesis have based their conclusions on the observed advances in a character in the same series and in "parallel" series. They overlook the fact that to-day there is experimental evidence demonstrating that variations as small even as those they record have been shown to rest on mutational stages. If the progress has been in the direction of adaptation, natural selection of small mutant differences will completely cover their findings. If it is claimed that in some of these cases the orthogenetic series is not in the line of adaptive advance, the burden of proof lies heavily on their shoulders. Moreover, the fact that recent work has made clear that genes generally have more than a single effect on the organization opens wide the door of suspicion, for the observed morphological progress might be a by-product of influences that have other and important, though unseen, effects. In a word, an orthogenetic series of changes does not in itself without a closer analysis than has as yet been furnished, establish that an *innate* *principle, urge, vis-a-tergo, or driving "force"* is causing the successive moves. The genetic evidence from multiple factors must create at least a strong suspicion against the "will to believe" in the mystic sentiments for which these terms always stand. That a progressive series of advances in a gene might take place with a consequent advance in the many characters involved is *thinkable,* especially if it could be shown that environmental changes cause parallel progress in the gene and this in turn in the character. How *probable* this is the reader must decide for himself in the light of the very clear evidence that each character is affected by changes in many genes differently located in the germ plasm and that it is not a progressive change in one gene that makes selection possible but changes in any one of many genes.

Chance Mutation and Natural Selection

The mutation process rests its argument for evolution on the view that among the possible changes in the genes, some combinations may happen to produce characters that are better suited to some place in the external world than were the original characters. Apparently this appeal to chance, like Darwin's appeal, has offended some sentimental adherents of the doctrine of organic evolution, because it has seemed to them inconceivable that chance could ever bring about the assembling of such an intricate piece of machinery as a highly complex organism. The attempt to mitigate the rude shock of the appeal to chance was made by Darwin by pointing out that evolution had been gradual and that the assemblage has not taken place out of chaos, but each stage has been built up on one a little less complex than the preceding one. Nevertheless, the fact remains that persistent efforts continue to be made from time to time to introduce into the theory of evolution some sort of directive agent. The Lamarckian theory has tried to bring about a more immediate relation between the organism and its environment of such a kind that the adaptive change that appears in the body as a result of a reaction between the environment and the animal or plant is reflected into the germ plasm. Bergson has cut the knot by postulating an innate adaptive responsiveness of the animal to every critical situation that calls out a response. The adherents of orthogenesis appeal, apparently—in so far as they commit themselves—to some sort of innate principle that causes advance in complexity along one line and they seem to hint at times even along directed lines of adaptation. Still more elusive are vague appeals made to some unknown principle—some sort of Mystical Element resident in living material and peculiar to it that is *Responsible* for evolution.

We are not concerned with any of these so-called "Laws" or Principles or Agents, but there is a relation between chance and evolution shown by living things that has been largely neglected, or at least vaguely referred to, even by natural selectionists, that is of fundamental importance when evolution is treated as a phenomenon of chance.

This relation may be stated in a general way as follows: Starting at any stage, the degree of development of any character increases the probability of further stages in the same direction. The relation can better be illustrated by specific cases. The familiar example of tossing pennies will serve. If I have thrown heads five times in succession, the chance that at the next toss of a penny I may make a run to six heads is greater than if I tossed six pennies at once. Not, of course, because five separate tosses of heads will increase the likelihood that at the next toss a head rather than a tail will turn up, but only that the chances are equal for a head or a tail, so that I have equal chances of increasing the run to six by that throw, while if I tossed six pennies at once the chances of getting six heads in one throw are only once in 64 times.

Similar illustrations in the case of animals and plants bring out the same point. If a race of men averages 5 feet 10 inches, and *on the average* mutations are not more than two inches above or below the racial average, the chance of a mutant individual appearing that is 6 feet tall is greater than in a race of 5-foot men. If increase in height is an advantage the taller race has a better chance than the smaller one. This statement does not exclude the possibility that a short race might *happen* to beat out in increment of growth a taller race, for it might more often mutate; but chance favors the tall. In this sense evolution is more likely to take place along the lines already followed, if further advantage is to be found in that direction.

A rolling snowball that already weighs 10 pounds is more likely to reach 15 pounds than is another that has just begun to roll. The chance that a monkey could change into a man is far greater than that ameba could make the transition. The monkey has accumulated, so to speak, so many of the things that go to make up a man that his chance of reaching that goal is vastly greater than ameba's.

There is also a peculiarity of animals and plants that assists greatly towards progress along lines already started. The individual multiplies itself, and a new mutant character that is advantageous becomes established in a large number of individuals, or even in all individuals of the race. The number of individuals increases the chance of a new random mutation along the path already taken. It is true that the number also increases the chance of a random variation in the opposite direction but as this, by hypothesis, is the less advantageous direction it will fail to establish itself in numbers.

ARTIFICIAL SELECTION AND MUTATION

Darwin built up his evidence for natural selection, and even for evolution, on the *artificial* selection of *variations* of animals and plants under domestication. It is in this field that the student of Mendelism revels. Almost without exception he finds that the domestic races of animals and plants are built up by mutational differences. It is this evidence that to-day is a hundredfold stronger for the theory of evolution than it was in Darwin's time. Shall we turn our back on it now when the evidence is clearer than ever before that the variations shown by domesticated animals and plants are in no way distinct from variations in the wild type?

The slightest familiarity with wild species will suffice to convince any one that they differ from each other generally not by a single Mendelian difference, but by a number of small differences. The student of Mendelian heredity at least is not likely to fall into the error of identifying single Mendelian differences with the sum total of differences by which wild types and often even wild varieties differ from each other. And whenever he has had an opportunity to study these single differences in wild varieties he has found that they seem to originate and to be inherited in the same way as other Mendelian characters.

To-day it is possible, in several groups where many small differences are known, to reconstruct races of individuals containing several of these small differences that any trained systematist, ignorant of their artificial origin, would classify as good species. To deny this shows only unfamiliarity with the genetic field; to admit it is to concede all that is claimed in favor of the theory that mutants may furnish the materials for selection.